

Positive T wave overshoot as a sign of ventricular enlargement

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SUMMARY A consecutive series of 86 patients with an inverted T wave showing terminal positivity (overshoot) of a specific pattern in the resting electrocardiogram were studied. Patients with bundle branch block or electrocardiographic evidence of acute infarction and those taking digoxin or a similar drug were excluded. In 67 patients the heart was examined by echocardiography and in a further two by direct inspection. Sixty six of the 69 patients had an abnormal thickness of the left (or right) ventricle or a calculated left ventricular mass >200 g. Seven of the patients examined by echocardiography had clinically pure ischaemic heart disease; all showed evidence of left ventricular enlargement. In only 39 of the 63 patients with anatomical evidence of left ventricular hypertrophy or dilatation did the electrocardiogram satisfy the standard voltage criterion of left ventricular hypertrophy.

In the absence of acute infarction, bundle branch block, or digitalisation positive T wave overshoot of the pattern described is a sign of increased ventricular mass.

Current electrocardiographic criteria for recognising left ventricular hypertrophy are seriously deficient. A considerable proportion of patients with repolarisation abnormalities due to left ventricular hypertrophy do not fulfil the standard voltage criterion of hypertrophy and the appearances are, therefore, assumed to indicate ischaemic heart disease.

In a study correlating the repolarisation pattern in V6 with the clinical diagnosis¹ the two features that showed the best correlation with diseases causing left ventricular hypertrophy (particularly hypertension and aortic valve disease) were pronounced asymmetry of the inverted T wave and terminal T wave positivity (overshoot). We have since shown that high grade asymmetry of the inverted T wave is strongly correlated with anatomical evidence of left (or right) ventricular hypertrophy or dilatation or an increase in calculated ventricular mass.² In the present study we aimed to correlate T wave overshoot with anatomical evidence of an increase in ventricular mass.

Patients and methods

DEFINING THE PATTERN OF OVERSHOOT

Overshoot is not confined to patients with left ven-

tricular hypertrophy. It is frequently seen in patients with ischaemic heart disease, especially during acute myocardial infarction and stress testing. The first need, therefore, is to examine the patterns of overshoot in patients with ischaemic heart disease and compare them with those seen in patients with diseases associated with left ventricular hypertrophy in order to determine whether they can be distinguished. If a pattern of overshoot can be defined that is apparently distinctive for patients with diseases associated with left ventricular enlargement a series of patients with such a pattern can then be studied by echocardiography to determine the state of the ventricle.

We, therefore, studied a group with ischaemic heart disease consisting of 51 patients (with 79 electrocardiograms) with myocardial infarction without clinical evidence of left ventricular hypertrophy from two previous studies^{1,3} and compared their electrocardiograms with those of a group with left ventricular hypertrophy, consisting of 75 patients (with 107 electrocardiograms) with aortic valve disease, hypertension, mitral regurgitation, or hypertrophic cardiomyopathy without clinical evidence of ischaemic heart disease from the same two studies.

As a result of this study, we were able to define a pattern of overshoot which was seen in the left ventricular hypertrophy group but not in the ischaemic heart disease group. The distinctive feature of this pattern was that the ST segment and descending limb

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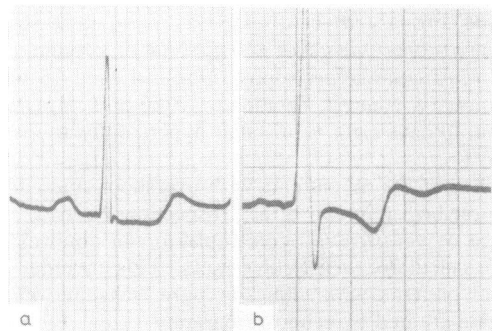


Fig. Electrocardiogram showing (a) non-specific overshoot in lead V6 in a woman aged 77 with meningioma and hypertension. (Neither echocardiography nor necropsy was performed.) (b) Left ventricular type overshoot in lead I in a man aged 47 with aortic regurgitation and mitral valve disease. Echocardiography showed posterior left ventricular wall thickness of 11 mm, interventricular septal thickness of 10 mm, and end diastolic diameter of 75 mm. The calculated left ventricular mass was 403 g. Note the upward convexity of the curve formed by the ST segment and the descending limb of the (inverted) T wave.

of the inverted T wave formed a curve which was convex upwards (Figure). Overshoot could be mimicked by a prominent upright U wave immediately after a prolonged inverted T wave.⁴ In our cases this possibility could usually be excluded with confidence either because the deflection fell within the duration of the T wave in other leads recorded at the same time or because of the presence of a distinct U wave after the overshoot.

STUDY POPULATION

Having defined a pattern of T wave positivity apparently characteristic of diseases associated with left ventricular hypertrophy, we studied a consecutive series of patients showing at least 1 mm of overshoot of this pattern in any lead of the resting electrocardiogram. We excluded patients with acute myocardial infarction or bundle branch block and those taking digoxin. Of the 86 patients identified, 52 were men and 34 women. Their ages ranged from 16 to 89 (mean 62) years.

INVESTIGATIONS

For each patient at least two 12 lead electrocardiograms were recorded. Overshoot was observed (or most pronounced) in lead V6 in 30 cases, in V5 in 19, in I in 14, in V4 in six, in aVF in five, in II in four, in aVL in three, in V2 in three, in V1 in one, and in V3 in one. The RS voltage in the precordial leads was measured by the method of Sokolow and Lyon.⁵ Each patient also had one departmental chest x ray exami-

nation performed. Twenty nine patients were studied by coronary arteriography and left heart catheterisation, and two by right heart catheterisation. In addition the hearts of two patients were inspected directly, one at operation and the other at necropsy.

A diagnosis of hypertension was based on two or more recorded systolic blood pressures of ≥ 200 mm Hg or diastolic blood pressures of ≥ 115 mm Hg in untreated patients or a systolic blood pressure of ≥ 160 mm Hg or a diastolic blood pressure of ≥ 100 mm Hg in patients taking antihypertensive agents. A diagnosis of ischaemic heart disease was based on the presence of either unequivocal evidence (historical, electrocardiographic, enzymatic, or necropsy) of myocardial infarction or of 70% narrowing in at least one main coronary artery.

In 39 cases overshoot was associated with a T wave asymmetry ratio of ≥ 2.0 in the same electrocardiogram, and in a further 11 cases with an asymmetry ratio of 2.0 in recording on another occasion. In 36 cases overshoot was not associated with an asymmetry ratio of ≥ 2.0 in any recording.

Echocardiography was performed on as many patients as possible. A few of the patients seen at the outpatient clinic lived too far from the hospital for it to be justified to bring them back for an investigation which would not affect their treatment. All the echocardiograms were performed by one investigator (JW) using an ATL Series 300 C with a paper speed of 50 mm/s. In one patient it was not possible to obtain a satisfactory recording. The remaining 67 echocardiograms were technically satisfactory in that adequate visualisation of the interventricular septum and posterior left ventricular wall were clearly defined just below the level of the free cusp edge of the mitral valve.

The wall thicknesses and the left ventricular intracavity diastolic dimension were measured at end diastole, being standardised to occur at the upstroke of the R wave of the electrocardiogram. The upper limit of normal for the interventricular septum and the posterior left ventricular wall was 12 mm and for the intracavity dimension at end diastole 56 mm. Left ventricular mass (LVM) was calculated using the Penn convention measurements (of left ventricular internal dimension (LVID), posterior wall thickness (PWT), and interventricular septal thickness (IVST)) in the equation $LVM = 1.04 [(LVID_p + PWT_p + IVST_p)^3 - (LVID_p)^3] - 13.6$ g.⁶ An upper limit of normal of 200 g was accepted.⁷

Results

Table 1 shows the clinical diagnosis in patients with overshoot and Table 2 the findings at echocardiography or on direct inspection.

Table 1 *Diagnostic analysis of 86 patients with overshoot*

Diagnosis	No of cases
Aortic valve disease	
Pure	23
Associated with ischaemic heart disease	3
Hypertension	
Pure	18
Associated with ischaemic heart disease	17
Mitral regurgitation, aortic valve disease, hypertension	9
Hypertrophic cardiomyopathy	4
Pure ischaemic heart disease	8
*Other	4
Total	86

*Two patients with pulmonary hypertension, one with pulmonary stenosis, and one with alcoholism.

Sixty seven patients were studied by echocardiography. In two, the overshoot was confined to leads V1 or V2. Both these patients had pulmonary hypertension, which in one case was associated with chronic lung disease and in the other case primary. Both showed right ventricular hypertrophy. The remaining 65 patients showed overshoot in one of the left ventricular leads. In 62 of these the calculated left ventricular mass was greater than 200 g.

Three patients with overshoot had no definite ventricular abnormality on echocardiography: a woman aged 49 years with aortic stenosis (gradient 50–60 mm Hg), mitral stenosis, and pulmonary hypertension; a woman aged 60 with a long history of hypertension in whom blood pressures of 180/90, 200/90, and 200/100 mm Hg were recorded while she was taking a combination of a beta blocking agent and a diuretic, and a man of 73 also with a long history of hypertension in whom two readings of 160/100 mm Hg were recorded while taking a beta blocking agent.

In two patients the heart was inspected directly. One man aged 84 with calcific aortic stenosis and

severe coronary artery disease was found at necropsy to have a heart weighing 650 g, showing pronounced left ventricular hypertrophy. One girl aged 16 with pulmonary stenosis (gradient 70–80 mm Hg) was found to have right ventricular hypertrophy at operation.

Thus of the 69 patients with overshoot studied by echocardiography or direct inspection 66 had evidence of ventricular hypertrophy or an abnormally large calculated ventricular mass. This includes all seven patients with clinically pure ischaemic heart disease studied by echocardiography.

The Sokolow and Lyon criterion of left ventricular hypertrophy⁵ was fulfilled in only 39 of the 63 patients with anatomical evidence of left ventricular hypertrophy. In 10 of the patients, the sum of S in V1 plus R in V5 or V6 was less than 30 mm.

Discussion

Terminal T wave positivity, or a "roller-coaster" repolarisation pattern, was noted in association with left ventricular hypertrophy in the early days of clinical electrocardiology,⁸ but the specificity of the pattern was never established. It is this that we have sought to do in the present study.

Terminal T wave positivity in general is not limited to left ventricular hypertrophy or dilatation. It is also seen in ischaemic heart disease and in patients who are taking digoxin. To differentiate the pattern seen in left ventricular enlargement from that seen in other conditions we analysed material from previous studies. We were able to define a pattern of terminal T wave positivity which was seen in a considerable proportion of patients with pure aortic valve disease, but in none of the patients with pure ischaemic heart disease. The distinctive feature of this pattern is an

Table 2 *Echocardiographic findings in 67 patients with overshoot*

Diagnosis	Total	Left ventricle				
		Wall thickness (>12 mm)	Diameter (>56 mm)	Wall thickness and diameter increased	Mass (>200 g)	No abnormality
Aortic valve disease						
Pure	16	11	1	4	15	0
Associated with ischaemic heart disease	2	1	1	0	2	0
Hypertension						
Pure	16	9	2	2	14	2
Associated with ischaemic heart disease	12	4	2	3	12	0
Mitral regurgitation, aortic valve disease, hypertension	8	2	2	3	7	1
Hypertrophic cardiomyopathy	4	3	1	0	3	0
Pure ischaemic heart disease	7	2	1	3	7	0
Other	2	RV+	RV+			0
Totals	67					

RV+, right ventricular hypertrophy present.

upward convexity of the ST segment and descending limb of the T wave (Figure).

In the present study, we compared this specific pattern of overshoot, observed in a consecutive series of patients, with the findings on echocardiography or direct observation. Of 69 patients studied by echocardiography or direct observation, in three the overshoot was confined to the right precordial leads. In each of these patients there was anatomical evidence of right ventricular hypertrophy or dilatation. In the remaining 66 patients, the overshoot was confined to one or more of the left ventricular leads. In 63 of these, there was evidence of either hypertrophy or increased diameter of the left ventricle or a calculated left ventricular mass of more than 200 g. Among these 66 patients, there were seven with clinically pure ischaemic heart disease. Each of these showed anatomical evidence of left ventricular enlargement. Of the three patients with overshoot who showed no echocardiographic evidence of left ventricular enlargement, one had significant aortic stenosis, one had severe hypertension, and one had borderline hypertension.

Twenty four of the 63 patients with anatomical evidence of left ventricular enlargement did not fulfil the Sokolow and Lyon⁵ voltage criterion of left ventricular hypertrophy and in 10 the sum of S in V1 and R in V5 or V6 was less than 30 mm. The deficiencies of the Sokolow and Lyon criterion are, of course, widely admitted; nevertheless it is still the generally accepted electrocardiographic criterion of left ventricular hypertrophy.⁹

Thus T wave overshoot of the pattern we have defined may be regarded as an additional independent

indicator of ventricular hypertrophy or dilatation.

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